AMENDMENTS TO THE CLAIMS

1. **(Currently Amended)** A method of improving renal function in a mammal in, or at risk of, chronic renal failure comprising administering to said mammal a therapeutically effective amount of a morphogen, said morphogen comprising an amino acid sequence having

   (a) at least 70% amino acid sequence homology with the C-terminal seven-cysteine skeleton of human OP-1, or

   (b) the sequence of the C-terminal seven-cysteine skeleton of human OP-1 being set forth at amino acids 330-431 of SEQ ID NO:1, or

   (c) at least 60% amino acid identity with the C-terminal seven-cysteine skeleton of human OP-1;

   wherein said mammal is afflicted with a chronic renal condition:

   (i) characterized by the progressive loss of renal function associated with the progressive loss of functioning nephron units; and

   (ii) comprising at least one of the following: chronic diabetic nephropathy, diabetic glomerulopathy, diabetic renal hypertrophy, hypertensive nephrosclerosis, hypertensive glomerulosclerosis, renal dysplasia, glomerular hypertrophy, tubular hypertrophy, glomerulosclerosis and tubulointerstitial sclerosis,

   wherein said morphogen induces chondrogenesis in an *in vivo* ectopic bone assay, and wherein said therapeutically effective amount causes a clinically significant improvement in a standard marker of renal function in said mammal, so as to thereby improve renal function in the mammal.

2. **(Currently Amended)** A method of delaying the need for, or reducing the frequency of, chronic dialysis treatments in a mammal in, or at risk of, chronic renal failure comprising administering to said mammal a therapeutically effective amount of a morphogen, said morphogen comprising an amino acid sequence having

   (a) at least 70% amino acid sequence homology with the C-terminal seven-cysteine skeleton of human OP-1, or

(b) the sequence of the C-terminal seven-cysteine skeleton of human OP-1 being set forth at amino acids 330-431 of SEQ ID NO:1, or

(c) at least 60% amino acid identity with the C-terminal seven-cysteine skeleton of human OP-1;

wherein said mammal is afflicted with a chronic renal condition:

(i) characterized by the progressive loss of renal function associated with the progressive loss of functioning nephron units; and

(ii) comprising at least one of the following: chronic diabetic nephropathy, diabetic glomerulopathy, diabetic renal hypertrophy, hypertensive nephrosclerosis, hypertensive glomerulosclerosis, renal dysplasia, glomerular hypertrophy, tubular hypertrophy, glomerulosclerosis and tubulointerstitial sclerosis,

wherein said morphogen induces chondrogenesis in an *in vivo* ectopic bone assay, and wherein said therapeutically effective amount causes a clinically significant improvement in a standard marker of renal function in said mammal, so as to thereby improve renal function in the mammal.

3. **(Previously Presented)** The method of claim 1, wherein said morphogen comprises a polypeptide comprising at least a C-terminal seven cysteine domain of a protein selected from the group consisting of OP-1, OP-2, OP-3, BMP2, BMP3, BMP4, BMP5, BMP6, BMP8, BMP9, GDF-5, GDF-6, GDF-7, DPP, Vg1, Vgr-1, 60A, GDF-1, GDF-3, GDF-5, GDF-6, GDF-7, BMP10, BMP13, BMP15, UNIVIN, NODAL, SCREW, ADMP, and NEURAL.

4. **(Previously Presented)** The method of claim 3, wherein said morphogen comprises a polypeptide consisting of at least a C-terminal seven cysteine domain of a protein selected from a group consisting of a pro form, a mature form, and a soluble form of human OP-1.

5. **(Canceled)**

6. **(Previously Presented)** The method of claim 1, wherein said morphogen has at least 75% homology with an amino acid sequence of a C-terminal seven-cysteine domain of human OP-1.

7. **(Previously Presented)** The method of claim 1, wherein said morphogen has at least 80% homology with an amino acid sequence of a C-terminal seven-cysteine domain of human OP-1 .

8. **(Previously Presented)** The method of claim 1, wherein said morphogen has at least 60% identity with an amino acid sequence of a C-terminal seven-cysteine domain of human OP-1 .

9. **(Previously Presented)** The method of claim 1, wherein said morphogen has at least 65% identity with an amino acid sequence of a C-terminal seven-cysteine domain of human OP-1 .

10. **(Previously Presented)** The method of claim 1, wherein said morphogen has at least 70% identity with an amino acid sequence of a C-terminal seven-cysteine domain of human OP-1 .

11. **(Canceled)**

12. **(Previously Presented)** The method of claim 1, wherein said morphogen is selected from the group consisting of OP-1, OP-2, OP-3, BMP2, BMP3, BMP4, BMP5, BMP6, BMP8, BMP9,GDF-5, GDF-6, GDF-7, DPP, Vgl, Vgr-1, 60A, GDF-1, GDF-3, GDF-5, GDF-6, GDF-7, BMP10, BMP13, BMP15, UNIVIN, NODAL, SCREW, ADMP, and NEURAL.

13-14. **(Canceled)**

15. **(Previously Presented)** The method of claim 1, wherein examination of said mammal indicates renal fibrosis.

16. **(Previously Presented)** The method of claim 15, wherein said examination is an ultrasound, MRI or CAT scan of said mammal.

17. **(Previously Presented)** The method of claim 1, wherein said mammal has less than about 50% of the functional nephron units of a mammal having intact healthy kidneys.

18-23. **(Canceled)**

24. **(Previously Presented)** The method of claim 1, wherein said mammal has a GFR which is chronically less than about 50% of a GFR_{exp} for said mammal.

25-27. **(Canceled)**

28. **(Previously Presented)** The method of claim 1, wherein said mammal is a human male weighing at least about 50 kg and has a GFR which is chronically less than about 50 ml/min.

29-31. **(Canceled)**

32. **(Previously Presented)** The method of claim 1, wherein said mammal is a human female weighing at least about 40 kg an has a GFR which is chronically less than about 40 ml/min.

33-51. **(Canceled)**

52. **(Previously Presented)** The method of claim 1, wherein said renal therapeutic agent is OP-1.

53. **(Previously Presented)** The method of claim 2, wherein said renal therapeutic agent is OP-1.

54-55. **(Canceled)**

56. **(Previously Presented)** The method of claim 1, wherein the morphogen is a dimeric polypeptide.

57. **(Previously Presented)** The method of claim 1, wherein the morphogen is a homodimer or a heterodimer.

58. **(Previously Presented)** The method of claim 2, wherein the morphogen is a dimeric polypeptide.

59. **(Previously Presented)** The method of claim 2, wherein the morphogen is a homodimer or a heterodimer.

60. **(New)** The method of claim 1, wherein the chronic renal condition is chronic diabetic nephropathy.

61. **(New)** The method of claim 1, wherein the chronic renal condition is diabetic glomerulopathy.

62. **(New)** The method of claim 1, wherein the chronic renal condition is diabetic renal hypertrophy.

63. **(New)** The method of claim 1, wherein the chronic renal condition is hypertensive nephrosclerosis.

64. **(New)** The method of claim 1, wherein the chronic renal condition is hypertensive glomerulosclerosis.

65. **(New)** The method of claim 1, wherein the chronic renal condition is renal dysplasia.

66. **(New)** The method of claim 1, wherein the chronic renal condition is glomerular hypertrophy.

67. **(New)** The method of claim 1, wherein the chronic renal condition is tubular hypertrophy.

68. **(New)** The method of claim 1, wherein the chronic renal condition is glomerulosclerosis.

69. **(New)** The method of claim 1, wherein the chronic renal condition is tubulointerstitial sclerosis